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Executive Summary

Exposure to environmental tobacco smoke (ETS) has been linked to a variety of adverse health outcomes. Many Californians are exposed at home, at work, and in public places. In the comprehensive reviews published as *Reports of the Surgeon General* and by the U.S. Environmental Protection Agency (U.S. EPA) and the National Research Council (NRC), ETS exposure has been found to be causally associated with respiratory illnesses—including lung cancer, childhood asthma, and lower respiratory tract infections. Scientific knowledge about ETS-related effects has expanded considerably since the release of the above-mentioned reviews. The state of California has therefore undertaken a broad review of ETS covering the major health endpoints potentially associated with ETS exposure: perinatal and postnatal manifestations of developmental toxicity, adverse impacts on male and female reproduction, respiratory disease, cancer, and cardiovascular disease. A "weight of evidence" approach has been used, in which the body of evidence is examined to determine whether or not it can be concluded that ETS exposure is causally associated with a particular effect. Because the epidemiological data are extensive, they serve as the primary basis for assessment of ETS-related effects in humans. The report also presents an overview on measurements of ETS exposure (particularly as they relate to characterizations of exposure in epidemiological investigations) and on the prevalence of ETS exposure in California and nationally.

ETS, or "secondhand smoke," is the complex mixture formed from the escaping smoke of a burning tobacco product and smoke exhaled by the smoker. The characteristics of ETS change as it ages and combines with other constituents in the ambient air. Exposure to ETS is also frequently referred to as "passive smoking," or "involuntary tobacco smoke" exposure. Although all exposures of the fetus are "passive" and "involuntary," for the purposes of this review, *in utero* exposure resulting from maternal smoking during pregnancy is not considered to be ETS exposure.

GENERAL FINDINGS

ETS is an important source of exposure to toxic air contaminants indoors. There is also some exposure outdoors in the vicinity of smokers. Despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures in the home, especially of infants and children, continue to be a public health concern. ETS exposure is causally associated with a number of health effects. Listed in Table ES.1 are the developmental, respiratory, carcinogenic, and cardiovascular effects for which there is sufficient evidence of a causal relationship—including fatal outcomes such as sudden infant death syndrome and heart disease

Table ES.1
Health Effects Associated with Exposure to Environmental Tobacco Smoke

Effects Causally Associated with ETS Exposure

Developmental Effects
Fetal Growth: Low birthweight or small for gestational age
Sudden Infant Death Syndrome (SIDS)

Respiratory Effects
Acute lower respiratory tract infections in children
(*e.g.*, bronchitis and pneumonia)
Asthma induction and exacerbation in children
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children

Carcinogenic Effects
Lung Cancer
Nasal Sinus Cancer

Cardiovascular Effects
Heart disease mortality
Acute and chronic coronary heart disease morbidity

Effects with Suggestive Evidence of a Causal Association with ETS Exposure

Developmental Effects
Spontaneous abortion
Adverse impact on cognition and behavior

Respiratory Effects
Exacerbation of cystic fibrosis
Decreased pulmonary function

Carcinogenic Effects
Cervical cancer

mortality, as well as serious chronic diseases such as childhood asthma. There are, in addition, effects for which evidence is suggestive of an association, but further research is needed for confirmation. These include spontaneous abortion, cervical cancer, and exacerbation of asthma in adults (Table ES.1). Finally, it is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints including congenital malformations, changes in female fertility and fecundability, male reproductive effects, rare childhood cancers, and cancers of the bladder, breast, stomach, brain, hematopoietic system, and lymphatic system.

[ii]**Author:** California Environmental Protection Agency.
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Many Californians are exposed to ETS, and the number of people adversely affected may be correspondingly large. Table ES.2 presents morbidity and mortality estimates for health effects causally associated with ETS exposure. For cancer, cardiovascular, and some respiratory endpoints, estimates are derived from figures published for the U.S. population, assuming that the number affected in California would be 12 percent of the total. The estimates for middle ear infection, sudden infant death syndrome, and low birthweight were derived using information on prevalence of ETS exposure in California and the U.S.

Relative risk estimates (RR) associated with some of these endpoints are small, but because the diseases are common, the overall impact can be quite large. A relative risk estimate of 1.3 for heart disease mortality in nonsmokers is supported by the collective evidence; this estimate corresponds to a lifetime risk of death of roughly 1 to 3 percent for exposed nonsmokers and approximately 4,000 deaths annually in California. The relative risk estimate of 1.2 to 1.4 associated with low birthweight implies that ETS may impact fetal growth of 1,200 to 2,200 newborns in California, roughly 1 to 2 percent of newborns of nonsmokers exposed at home or at work. ETS may exacerbate asthma (RR $\approx$ 1.6 to 2) in 48,000 to 120,000 children in California. Large impacts are associated with relative risks for respiratory effects in children such as middle ear infection (RR $\approx$ 1.62) and lower respiratory disease in young children (RR $\approx$ 1.5 to 2). Asthma induction (RR $\approx$ 1.75 to 2.25) may occur in as many as 0.5 to 2 percent of ETS-exposed children. ETS exposure may be implicated in 120 SIDS deaths per year in California (RR $\approx$ 3.5), with a risk of death approaching 0.1 percent for infants exposed to ETS in their homes. Lifetime risk of lung cancer death related to ETS-exposed nonsmokers may be about 0.7 percent (RR $\approx$ 1.2). For nasal sinus cancers, observed relative risks have ranged from 1.7 to 3.0, but future studies are needed to confirm the magnitude of ETS-related risks.

SPECIFIC FINDINGS AND CONCLUSIONS

Exposure Measurement and Prevalence

ETS is a complex mixture of chemicals generated during the burning and smoking of tobacco products. Chemicals present in ETS include irritants and systemic toxicants such as hydrogen cyanide and sulfur dioxide; mutagens and carcinogens such as benzo[a]pyrene, formaldehyde, and 4-aminobiphenyl; and the reproductive toxicants nicotine, cadmium, and carbon monoxide. Many ETS constituents have been identified as hazardous by state, federal, and international agencies. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six identified as developmental or reproductive toxicants under California's Proposition 65 (California Health and Safety Code 25249.5 *et seq.*).

Exposure assessment is critical in epidemiological investigations of the health impacts of ETS, and in evaluating the effectiveness of strategies to reduce exposure. Exposure can be assessed through the measurement of indoor air concentrations of ETS constituents, through surveys and ques-

Exposure Measurement and Prevalence

2.1 INTRODUCTION This chapter provides background information on the prevalence and measurement of exposure to ETS and emphasizes investigation and monitoring methods used in epidemiological evaluations of health effects. Section 2.2 briefly reviews the physical and chemical properties of ETS and identifies some of the important biologically active constituents present in ETS. Section 2.3 discusses various techniques that have been used to measure ETS concentrations in indoor environments. Determination of ETS contamination is a challenge, as ETS is a complex mixture of over 4,000 compounds, and it is neither feasible nor practical to characterize every individual constituent of ETS. Given the complex nature of ETS, markers and tracers of ETS are measured to assess ETS exposures. The role and limitations of some ETS markers, such as nicotine, particulate matter in air, and polycyclic aromatic hydrocarbons, are discussed in this section. Section 2.4 addresses the use of biomarkers to measure ETS exposure. In addition to being dependant on ETS concentration in air, the measured level of biomarker varies with an individual's uptake, distribution, metabolism, and excretion of the chemical of interest. This section describes the use and limitations of some of the biomarkers, such as nicotine and cotinine in physiological fluids, in determining ETS exposure.

One problem with ETS markers and biomarkers is that most of them are only capable of estimating ETS exposure over a relatively short period of time, from a few hours to several weeks, whereas many health effects of ETS are believed to be associated with long-term exposures that are measured in months, if not years. In order to address this difficulty, most epidemiological studies cited in this report used questionnaires or interviews to determine the status of the subjects regarding long-term exposure to ETS. Some studies also used measurements of ETS markers and biomarkers as supplemental information. And just like any epidemiological study that relies on questionnaires or interviews for exposure information, these studies are subjected to the problem of misclassification. Section 2.5 of this chapter describes some of the difficulties associated with classifying subjects into exposure categories based on the smoking status of other household members. As of today, no perfect method for quantifying ETS exposure has been found. Yet, as demonstrated by many studies cited in other chapters of the report, epidemiologists are able to use the information obtained from questionnaires or interviews in classifying the subjects into categorical groups of ETS exposure (*e.g.*, none, low, medium, or high). The categorical exposure information is then used to evaluate health risks associated with ETS exposure. However, one drawback of this approach is that it decreases the sensitivity or power of a study—*i.e.*, it will not show a positive association when a health effect is only moderately related to ETS exposure.

Though many ETS monitoring methods (*e.g.*, nicotine and respirable suspended particulates in air, cotinine in body fluids) are discussed in this chapter, risk assessment of ETS exposure is seldom performed based on monitoring results. Some of the reasons include short sampling duration in most studies, large uncertainty in extrapolating the ETS levels measured at a specific location to the general population, and large uncertainty in estimating the frequency and duration of ETS exposure of the general population. Consistent with the approach used by the National Research Council (NRC, 1986), U.S. EPA (1992), DiFranza and Lew (1996), and Wells (1994), this report uses prevalence assessment for the estimation of health risks that are associated with past or recent ETS exposure. Epidemiologists often use prevalence assessment, which makes use of semi-quantitative exposure information, such as job classification or duration of exposure, for the estimation of health risks associated with occupational and environmental hazards.

Section 2.6 discusses the prevalence of ETS exposures and factors affecting prevalence, especially in California. In support of the assessment of reproductive and developmental effects presented in the chapters addressing these effects, information on both measurement and prevalence of ETS exposures of the developing child (*in utero*, during infancy, and during childhood) is described when available.

2.2 PROPERTIES OF ETS AND ITS CONSTITUENTS

2.2.1 Physical and Chemical Properties of ETS[1]

ETS is a complex mixture of chemicals generated during the burning of tobacco products. The principal contributor to ETS is "sidestream smoke," the material emitted from the smoldering tobacco product between puffs. Other components of ETS include exhaled mainstream smoke, mainstream smoke emitted at the mouthpiece during puff drawing, and compounds diffused through the wrapper. "Mainstream smoke" is the complex mixture that exits from the mouthpiece of a burning cigarette when a puff is inhaled by the smoker.

When a cigarette is smoked, approximately one-half or more of the smoke generated (by weight) is sidestream smoke emitted from the smoldering cigarette. The chemical composition of mainstream smoke has been more extensively characterized than that of sidestream smoke, but they are produced by the same fundamental processes, such that many chemical constituents are present in both. Over 4,000 individual constituents have been identified in mainstream smoke, and approximately 400 compounds have been measured quantitatively in both mainstream and sidestream smoke.

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1 The U.S. EPA (1992) report is the primary source of information presented in this section; unless a specific reference is provided, the information in this section has been taken from that report.

The large number of constituents results from the chemical composition of tobacco and the variety of chemical and physical processes that occur as a cigarette is smoked. The majority of the compounds present in mainstream smoke are formed during combustion, in a pyrolysis-distillation zone just behind the heat-generating combustion zone (Baker, 1981). Estimates have been made that the total number of constituents in mainstream smoke actually may be 10 to 20 times the number identified to date; that is, mainstream smoke may comprise over 100,000 constituents. However, these unidentified components comprise less than 5 percent of the mass of mainstream smoke and would be present only at very low concentrations (Guerin *et al.*, 1992).

Although many constituents present in mainstream and sidestream smoke are the same, there are important differences in their rates of emission into the air due to physical and chemical differences in the burning conditions present during their generation. As discussed in *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* (U.S. EPA, 1992: pages 3-2 to 3-10), some constituents have a higher rate of release into sidestream than mainstream smoke, while for others the reverse is true. Once emitted into the air, sidestream smoke may undergo various physical and chemical changes. Dilution, chemical reactions, deposition, and other removal processes may decrease the concentration of the airborne constituents of ETS, alter the size distribution of suspended particles, and chemically modify some of the more reactive constituents of ETS.

The delivery of selected agents in the mainstream smoke of nonfilter cigarettes and the ratios of the relative distribution of these agents in sidestream to mainstream smoke are given in U.S. EPA (1992: Table 3-1). As discussed by U.S. EPA (1992: pages 3-4 to 3-6), sidestream to mainstream ratios are highly variable and can be misleading, as a number of factors affecting cigarette design (*e.g.*, presence of a filter and filter ventilation) and smoking patterns (*e.g.*, puff volume) have a substantial impact on the emissions of mainstream smoke. In contrast, sidestream smoke emissions show relatively little variability as a function of most of these same factors. A study of the influence of puff volume and filter ventilation on sidestream and mainstream deliveries illustrates this point (Browne *et al.*, 1980). The mainstream delivery of particulate matter and carbon monoxide increases with puff volume, but decreases with increasing filter ventilation. Because the sidestream delivery of these constituents remains relatively constant, the corresponding sidestream to mainstream ratios will decrease or increase as a function of the specific condition and constituent examined (Table 2.1).

Data on sidestream emission rates from filtered and commercial cigarettes for many compounds of public health interest are tabulated in U.S. EPA (1992: Table 3-2). While the data are limited, they suggest that sidestream deliveries are relatively constant across a number of products, with differences ranging two- to three-fold when measured under standard smoking conditions. These results are consistent with the finding that side-

Table 2.1

Influence of Puff Volume and Filter Ventilation on Deliveries of Particulate Matter and Carbon Monoxide in Mainstream and Sidestream Smoke

		Milligrams per Cigarette and SS/MS ratio					
	# of	Particulate Matter			Carbon Monoxide		
Variable[a]	Puffs	MS	SS	SS/MS	MS	SS	SS/MS
Puff Volume							
None, Free burn	0	--	23	--	--	58	--
17.5 cc	9.6	29	23	0.8	9	63	7
35 cc	8.7	46	20	0.4	19	50	2.6
50 cc	7.4	55	21	0.4	20	56	2.8
Filter Ventilation[b]							
0%	8.7	46	20	0.4	19	50	2.6
33%	8.8	32	21	0.6	13	49	3.8
48%	9.8	21	21	1.0	7	58	8.3
83%	10.6	12	21	1.8	2	56	2.8

Browne et al. (1980)

[a] *USA blend cigarette, FTC smoking conditions unless otherwise noted.*
[b] *Percentage of mainstream puff air entering through periphery of filter.*

stream deliveries are primarily related to the weight of the tobacco and paper consumed during smoldering, rather than to cigarette design (Guerin *et al.*, 1992).

2.2.2 Biologically Active Constituents of ETS

A number of chemicals known or suspected to contribute to adverse health effects are present in tobacco smoke (mainstream and sidestream smoke), including eye and respiratory irritants, systemic toxicants, mutagens, carcinogens, and reproductive toxicants. It is outside the scope of this review to assess exposure to each of the numerous individual constituents of ETS or their specific contribution to the health effects associated with ETS. This section provides a brief discussion of some of the more toxicologically significant compounds identified in tobacco smoke.

2.2.2.1 Toxicants with Acute Effects

Irritants and toxicants with other acute health effects have been identified in ETS, including ammonia, acrolein, carbon monoxide, formaldehyde, hydrogen cyanide, nicotine, nitrogen oxides, phenol, and sulfur dioxide. Ammonia, formaldehyde, and sulfur dioxide are respiratory irritants and may exacerbate the condition of people with breathing difficulties. Several components, including acrolein, crotonaldehyde, formaldehyde, and hydrogen cyanide, affect mucociliary function, and at a sufficiently high concentration can inhibit clearance of smoke par-

Table 2.2

Chemical Constituents of Tobacco Smoke That Have Been Classified or Identified as to their Carcinogenicity, Reproductive Toxicity, or Other Health Hazard

COMPOUND	IARC Classification[a]	U.S. EPA Classification[b]	CAL/EPA Prop 65[c]//TAC[d]
Organic Compounds			
Acetaldehyde	2B	B2	yes//yes
Acetamide	2B		yes//yes
Acrolein	3	C	--- //yes
Acrylonitrile	2A	B1	yes//yes
4-Aminobiphenyl	1		yes//yes
Aniline	3	B2	yes//yes
o-Anisidine	2B		yes//yes
Benz[a]anthracene	2A	B2	yes//yes
Benzene	1	A	yes//yes
Benzo[b]fluoranthene	2B	B2	yes//yes
Benzo[j]fluoranthene	2B		yes//yes
Benzo[k]fluoranthene	2B	B2	yes//yes
Benzo[a]pyrene	2A	B2	yes//yes
1,3-Butadiene		B2	yes//yes
Captan	3		yes//yes
Carbon disulfide[e]			yes//yes
Carbon monoxide[e]			yes//---
Chrysene	3	B2	yes//yes
DDT	2B		yes//---
Dibenz[a,h]acridine	2B		yes//yes
Dibenz[a,j]acridine	2B		yes//yes
Dibenz[a,h]anthracene	2A	B2	yes//yes
7H-Dibenzo[c,g]carbazole	2B		yes//yes
Dibenzo[a,e]pyrene	2B		yes//yes
Dibenzo[a,h]pyrene	2B		yes//yes
Dibenzo[a,i]pyrene	2B		yes//yes
Dibenzo[a,l]pyrene	2B		yes//yes
1,1-Dimethylhydrazine	2B		yes//yes
1-Naphthylamine	3		yes//---
2-Naphthylamine	1		yes//---
Nicotine[e]			yes//---
2-Nitropropane	2B		yes//yes
N-Nitrosodi-n-butylamine	2B	B2	yes//---
N-Nitrosodiethanolamine	2B	B2	yes//---
N-Nitrosodiethylamine	2A	B2	yes//---
N-Nitroso-n-methylethylamine	2B	B2	yes//---
N'-Nitrosonornicotine	2B		yes//---
N-Nitrosopiperidine	2B		yes//---
N-Nitrosopyrrolidine	2B		---//yes
Styrene	2B		---//yes
Toluene[e]			yes//yes
2-Toluidine	2B		yes//yes
Urethane	2B		yes//---
Vinyl chloride	1		yes//yes

Table 2.2 (*Continued*)

COMPOUND	IARC Classification[a]	U.S. EPA Classification[b]	CAL/EPA Prop 65[c]/TAC[d]
Inorganic Compounds			
Arsenic	1	A	yes//yes
Cadmium	2A	B1	yes//yes
Chromium V1	1	A	yes//yes
Lead[e]	2B	B2	yes//yes
Nickel	1	A	yes//yes

Sources: ARB (1993); IARC (1985, 1986, 1987, 1992); California Code of Regulations (1994); U.S. EPA (1994)

[a] *International Agency for Research on Cancer (IARC) Classification: 1, carcinogenic to humans; 2A, probably carcinogenic to humans; 2B, possibly carcinogenic to humans; 3, not classifiable as to its carcinogenicity to humans.*

[b] *U.S. EPA Classification: A, human carcinogen; B1, probable human carcinogen (primarily on the basis of epidemiological data); B2, probable human carcinogen (primarily on the basis of animal data); C, possible human carcinogen.*

[c] *Chemicals listed under Proposition 65 are known to the State to cause cancer or reproductive toxicity (California Health and Safety Code Section 25249.5 et seq.).*

[d] *Substances identified as Toxic Air Contaminants by the Air Resources Board (ARB), pursuant to the provisions of AB 1807 and AB 2728 (includes all Hazardous Air Pollutants listed in the Federal Clean Air Act Amendments of 1990).*

[e] *Reproductive toxicant*

ticles from the lung (Battista, 1976). Nicotine, which is the principal alkaloid in tobacco, is a major contributor to the addictive properties of tobacco. Nicotine has diverse pharmacologic and toxicological actions, ranging from acute poisoning to chronic effects, some of which may be responsible for some of the adverse health effects associated with smoking.

2.2.2.2 Toxicants with Carcinogenic Effects

Over 50 compounds have been identified in tobacco smoke that are recognized as known or probable human carcinogens. These compounds, which may occur naturally in tobacco or which are formed during combustion, reside mainly in the particulate phase (IARC, 1986). Most of the major classes of carcinogens, including both organic and inorganic constituents, are represented. Table 2.2 lists those compounds detected in tobacco smoke for which there is evidence of animal or human carcinogenicity, as evaluated by the U.S. EPA or the IARC. Also in Table 2.2 are compounds listed as carcinogens under California's Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65, California Code of Regulations, Title 22, Section 12000) and a number of tobacco smoke constituents that have been identified as toxic air contaminants by the California Air Resources Board (ARB, 1993). Tobacco smoke itself is listed as a carcinogen under Proposition 65.

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Table ES.2
Estimated Annual Morbidity and Mortality in Nonsmokers Associated with ETS Exposure

Condition	Number of People or Cases[a] in the U.S.	in California
Developmental Effects		
Low birthweight	9,700 - 18,600 cases[b]	1,200 - 2,200 cases[b]
Sudden Infant Death Syndrome (SIDS)	1,900 - 2,700 deaths[b]	120 deaths[b]
Respiratory Effects in Children		
Middle ear infection	0.7 to 1.6 million physician office visits[b]	78,600 to 188,700 physician office visits[b]
Asthma induction	8,000 to 26,000 new cases[c]	960 to 3,120 new cases[c]
Asthma exacerbation	400,000 to 1,000,000 children[c]	48,000 to 120,000 children[c]
Bronchitis or pneumonia in infants and toddlers (18 months and under)	150,000 to 300,000 cases[c] 7,500 to 15,000 hospitalizations[c] 136 - 212 deaths[c]	18,000 to 36,000 cases[c] 900 to 1,800 hospitalizations[c] 16 - 25 deaths[c]
Cancer		
Lung	3,000 deaths[c]	360 deaths[c]
Nasal sinus	N/A[d]	N/A[d]
Cardiovascular Effects		
Ischemic heart disease	35,000 - 62,000 deaths[c]	4,200 - 7,440 deaths[c]

[a] *The numbers in the table are based on maximum likelihood estimates of the relative risk. As discussed in the body of the report, there are uncertainties in these estimates, so actual impacts could be somewhat higher or lower than indicated in the table. The endpoints listed are those for which there is a causal association with ETS exposure based on observations of effects in exposed human populations.*

[b] *California estimates for low birthweight, SIDS, and middle ear infection (otitis media) are provided in Chapters 3, 4, and 6, respectively. U.S. estimates are obtained by dividing by 12 percent, the fraction of the U.S. population residing in California.*

[c] *Estimates of mortality in the U.S. for lung cancer and respiratory effects, with the exception of middle ear infection (otitis media), come from U.S. EPA (1992). U.S. range for heart disease mortality reflects estimates reported in Wells (1988 and 1994), Glantz and Parmley (1991), Steenland (1992). California predictions are made by multiplying the U.S. estimate by 12 percent, the fraction of the U.S. population residing in the State. Because of decreases in smoking prevalence in California in recent years, the number of cases for some endpoints may be somewhat overestimated, depending on the relative impacts of current versus past ETS exposures on the health endpoint.*

[d] *Estimates of the impact of ETS exposure on the occurrence of nasal sinus cancers are not available at this time.*

Most of the ETS population impact estimates are presented in terms of ranges, which are thought to reflect reasonable assumptions about the estimates of parameters and variables required for the extrapolation models. The validity of the ranges is also dependent on the appropriateness of the extrapolation models themselves.

While this report focuses only on the respiratory health effects of passive smoking, there also may be other health effects of concern. Recent analyses of more than a dozen epidemiology and toxicology studies (e.g., Steenland, 1992; National Institute for Occupational Safety and Health [NIOSH], 1991) suggest that ETS exposure may be a risk factor for cardiovascular disease. In addition, a few studies in the literature link ETS exposure to cancers of other sites; at this time, that database appears inadequate for any conclusion. This report does not develop an analysis of either the nonrespiratory cancer or the heart disease data and takes no position on whether ETS is a risk factor for these diseases. If it is, the total public health impact from ETS will be greater than that discussed here.

1.3. PRIMARY FINDINGS

A. Lung Cancer in Nonsmoking Adults

1. Passive smoking is causally associated with lung cancer in adults, and ETS, by the total weight of evidence, belongs in the category of compounds classified by EPA as Group A (known human) carcinogens.
2. Approximately 3,000 lung cancer deaths per year among nonsmokers (never-smokers and former smokers) of both sexes are estimated to be attributable to ETS in the United States. While there are statistical and modeling uncertainties in this estimate, and the true number may be higher or lower, the assumptions used in this analysis would tend to underestimate the actual population risk. The overall confidence in this estimate is medium to high.

B. Noncancer Respiratory Diseases and Disorders

1. Exposure of children to ETS from parental smoking is causally associated with:
    a. increased prevalence of respiratory symptoms of irritation (cough, sputum, and wheeze),
    b. increased prevalence of middle ear effusion (a sign of middle ear disease), and
    c. a small but statistically significant reduction in lung function as tested by objective measures of lung capacity.
2. ETS exposure of young children and particularly infants from parental (and especially mother's) smoking is causally associated with an increased risk of LRIs (pneumonia, bronchitis, and bronchiolitis). This report estimates that exposure to ETS contributes 150,000 to 300,000 LRIs annually in infants and children less than 18 months of age, resulting in 7,500 to 15,000 hospitalizations. The confidence in the estimates of LRIs is high. Increased risks for LRIs continue, but are lower in magnitude, for children until about age 3; however, no estimates are derived for children over 18 months.

1.85), the estimated relative risks are higher than those of the United States and more highly significant after adjusting for the potential bias.

- Strong associations for highest exposure groups. Examining the groups with the highest exposure levels increases the ability to detect an effect, if it exists. Nine of the sixteen studies worldwide for which there are sufficient exposure-level data are statistically significant for the highest exposure group, despite most having low statistical power. The overall pooled estimate of 1.81 for the highest exposure groups is highly statistically significant (90% C.I. = 1.60, 2.05; $p < 10^{-6}$). For the United States, the overall pooled estimate of 1.38 (seven studies, corrected for smoker misclassification bias) is also highly statistically significant (90% C.I. = 1.13, 1.70; $p = 0.005$).
- Confounding cannot explain the association. The broad-based evidence for an association found by independent investigators across several countries, as well as the positive exposure-response trends observed in most of the studies that analyzed for them, make any single confounder highly unlikely as an explanation for the results. In addition, this report examined potential confounding factors (history of lung disease, home heat sources, diet, occupation) and concluded that none of these factors could account for the observed association between lung cancer and ETS.

1.3.1.2. *Estimation of Population Risk*

The individual risk of lung cancer from exposure to ETS does not have to be very large to translate into a significant health hazard to the U.S. population because of the large number of smokers and the widespread presence of ETS. Current smokers comprise approximately 26% of the U.S. adult population and consume more than one-half trillion cigarettes annually (1.5 packs per day, on average), causing nearly universal exposure to at least some ETS. As a biomarker of tobacco smoke uptake, cotinine, a metabolite of the tobacco-specific compound nicotine, is detectable in the blood, saliva, and urine of persons recently exposed to tobacco smoke. Cotinine has typically been detected in 50% to 75% of reported nonsmokers tested (50% equates to 63 million U.S. nonsmokers age 18 or older).

The best estimate of approximately 3,000 lung cancer deaths per year in U.S. nonsmokers age 35 and over attributable to ETS (Chapter 6) is based on data pooled from all 11 U.S. epidemiologic studies of never-smoking women married to smoking spouses. Use of U.S. studies should increase the confidence in these estimates. Some mathematical modeling is required to adjust for expected bias from misclassification of smoking status and to account for ETS exposure from sources other than spousal smoking. The overall relative risk estimate of 1.19 for the United States, already adjusted for smoker misclassification bias, becomes 1.59 after adjusting for background ETS sources (1.34 for nonspousal exposures only). Assumptions are also needed to relate responses in female never-smokers to those in male never-smokers and ex-smokers of both sexes, and to estimate the proportion of the nonsmoking population exposed to various levels of ETS. Overall, however, the assumptions necessary for estimating risk add far less uncertainty than other EPA quantitative assessments. This is because the extrapolation for

ETS is based on a large database of human studies, all at levels actually expected to be encountered by much of the U.S. population.

The components of the 3,000 lung cancer deaths figure include approximately 1,500 female never-smokers, 500 male never-smokers, and 1,000 former smokers of both sexes. More females are estimated to be affected because there are more female than male nonsmokers. These component estimates have varying degrees of confidence; the estimate of 1,500 deaths for female never-smokers has the highest confidence because of the extensive database. The estimate of 500 for male never-smokers is less certain because it is based on the female never-smoker response and is thought to be low because males are generally subject to higher background ETS exposures than females. Adjustment for this higher background exposure would lead to higher risk estimates. The estimate of 1,000 lung cancer deaths for former smokers of both sexes is considered to have the lowest confidence, and the assumptions used are thought to make this estimate low as well.

Workplace ETS levels are generally comparable with home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that nonspousal exposures to ETS are often greater than exposure from spousal smoking. Thus, this report presents an alternative breakdown of the estimated 3,000 ETS-attributable lung cancer deaths between spousal and nonspousal exposures. By extension of the results from spousal smoking studies, coupled with biological measurements of exposure, more lung cancer deaths are estimated to be attributable to ETS from combined nonspousal exposures--2,200 of both sexes--than from spousal exposure--800 of both sexes. This spouse-versus-other-sources partitioning depends on current exposure estimates that may or may not be applicable to the exposure period of interest. Thus, this breakdown contains this element of uncertainty in addition to those discussed above with respect to the previous breakdown.

An alternative analysis, based on the large Fontham et al. (1991) study, which is the only study that provides biomarker estimates of both relative risk and ETS exposure, yields population risk point estimates of 2,700 and 3,600. These population risk estimates are highly consistent with the estimate of 3,000 based on the combined U.S. studies.

While there is statistical variance around all of the parameters used in the quantitative assessment, the two largest areas of uncertainty are probably associated with the relative risk estimate for spousal ETS exposure and the parameter estimate for the background ETS exposure adjustment. A sensitivity analysis that independently varies these two estimates yields population risk estimates as low as 400 and as high as 7,000. These extremes, however, are considered unlikely; the more probable range is narrower, and the generally conservative assumptions employed suggest that the actual population risk number may be greater than 3,000. Overall, considering the multitude, consistency, and quality of all these studies, the weight-of-evidence conclusion that ETS is a known human lung carcinogen, and the limited amount of extrapolation necessary, the confidence in the estimate of approximately 3,000 lung cancer deaths is medium to high.

[iv] **Author:** Taylor, A., Johnson, D, & Kazemi, H.
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AHA Medical/Scientific Statement

Position Statement

Environmental Tobacco Smoke and Cardiovascular Disease

A Position Paper From the Council on Cardiopulmonary and Critical Care, American Heart Association

Aubrey E. Taylor, PhD, Chairman; Douglas C. Johnson, MD, and Homayoun Kazemi, MD, Members

Cigarette smoking was identified by the Surgeon General in 1982 and 1983 as the most important modifiable risk factor for cancer and chronic heart disease in the United States.[1,2] Recent studies have implicated exposure to environmental tobacco smoke as a significant risk factor for the development of lung cancer and heart disease. Because more information on environmental tobacco smoke is now available, its health effects are reviewed in this report, with a major emphasis on the relation of environmental tobacco smoke to cardiovascular disease.

Cigarette smoking has a significant effect on the health of Americans, and is a major cause of cardiovascular disease.[3] Cardiovascular disease attributable to voluntary cigarette smoking accounts for about as many deaths each year as chronic obstructive pulmonary disease and lung cancer deaths combined. In 1988 approximately 430,000 deaths in adults aged 35 and older were attributed to the intentional inhalation of tobacco smoke. This number included 201,000 deaths due to cardiovascular disease, 112,000 due to lung cancers, 83,000 due to chronic lung disease (including pneumonia, influenza, bronchitis, emphysema, chronic airway obstruction, and other respiratory diseases), and 31,000 due to other cancers.[4] It has also been estimated that an additional 3,800 lung cancer deaths[4] and 37,000 cardiovascular deaths occurred in nonsmokers who had been exposed to environmental tobacco smoke.[5] An additional 2,500 perinatal deaths were estimated to have occurred because of maternal smoking, and about 1,300 deaths resulted from burns related to smoking.[4]

Although the existing epidemiological studies on cancer deaths associated with environmental tobacco smoke may be subject to questions about sample size, exposure, experimental design, and differing lifestyles of populations, sufficient information has been published to implicate environmental tobacco smoke as a definite health hazard. The 1986 Surgeon General's report concluded that involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers, and it was postulated that approximately 3,000–4,000 nonsmokers exposed to environmental tobacco smoke die of lung cancer each year.[6] The report also concluded that children whose parents smoke have an increased frequency of respiratory infections, increased symptoms of respiratory problems, and slightly smaller rates of increase in lung function as the lung matures compared with children of nonsmoking parents. At the time of the report, environmental tobacco smoke could not be definitely linked to cardiovascular disease. However, since 1986 several studies have been published documenting a link between environmental tobacco smoke, cancer,[7] and heart disease.[5,8] The Environmental Protection Agency has also done an extensive study of the effects of environmental tobacco smoke on lung cancer.

Environmental Tobacco Smoke

Burning cigarettes emit two types of smoke: mainstream smoke, which is the smoke directly inhaled into the smoker's lungs, and sidestream smoke, which is the smoke emitted into the air from the burning cigarette between puffs. Environmental tobacco smoke is about 85% sidestream and 15% exhaled mainstream smoke. More than 4,000 chemicals, including at least 40 carcinogens, are contained in environmental tobacco smoke.[9] Many toxic constituents are found in higher concentrations in sidestream than in mainstream smoke.[5] For example, in sidestream smoke there is about five times as much carbon monoxide (which decreases the ability of hemoglobin to carry oxygen to the tissues), three times as much benzopyrene (a tumor- and plaque-producing compound), and 50 times as much ammonia (an eye and respiratory irritant) as is inhaled directly from a cigarette. The difference is because the cigarette burns at a higher temperature during inhalation, leading to more complete combustion, and filters also screen some of these toxic compounds.

Those in close proximity to someone smoking a cigarette are exposed to smoke not only while the cigarette is lit but continue to inhale smoke that has mixed with air long after the cigarette is extinguished. Environmental tobacco smoke can persist in indoor environments for many hours after cessation of smoking, the time depending on ventilation and the mixing of

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room air with uncontaminated air.[10] To conserve energy, building ventilation rates are sometimes decreased, causing levels of smoke to increase in workplace environments, and in many homes ventilation of smoke to the outside is minimal.

Risk to Nonsmokers from Environmental Tobacco Smoke

The relative risk of developing lung cancer has been estimated to be 1.3 for nonsmokers exposed to environmental tobacco smoke at home compared with nonsmokers with no exposure to environmental tobacco smoke.[7,10,12] Active smoking has a relative risk factor for cancer of about 10.[1] Average workplace exposures to environmental tobacco smoke are estimated to increase lung cancer risk twofold because environmental tobacco smoke exposures are generally higher at the workplace than at home.[12] Despite the difficulty of interpreting epidemiological studies of exposure levels in the home and workplace, several recent studies demonstrate a definite link between cardiovascular deaths in nonsmokers exposed to environmental tobacco smoke. Glantz and Parmley[5] reviewed 10 of these studies, showing that men and women nonsmokers exposed to environmental tobacco smoke at home had an overall cardiovascular relative risk factor of 1.3. This compares to a relative risk factor of 1.7 for smokers compared with nonsmokers.[2] Kawachi et al[13] predicted an even higher relative risk factor for workplace exposures of nonsmokers to environmental tobacco smoke.

Repace and Lowrey[6] evaluated eight studies in which the number of lung cancer deaths of nonsmokers exposed to environmental tobacco smoke averaged 5,000±2,400 (mean±standard deviation) per year. Assuming that the ratio of lung cancer to heart disease deaths is the same with environmental tobacco smoke exposure as for voluntary smoking, approximately 10,000 deaths of nonsmokers exposed to environmental tobacco smoke would be expected to occur per year. However, this simple estimate does not include many aspects of environmental tobacco smoke exposure, such as the amount of environmental tobacco smoke exposure in the workplace and home, the number of persons exposed to environmental tobacco smoke, and the type and amount of smoke exposure. In fact, studies to evaluate these factors indicate that environmental tobacco smoke causes a higher risk of heart disease than predicted by this simple estimate.

Recently, Steenland[8] performed extensive analyses of the available literature on the cardiovascular effects of environmental tobacco smoke and predicted that ischemic heart disease could cause as many as 15,000–19,000 deaths yearly of nonsmokers due solely to environmental tobacco smoke from their spouses. Steenland also predicted an overall number of deaths due to environmental tobacco smoke–related cardiovascular disease of 35,000–40,000 yearly, a number similar to the number of deaths estimated by Glantz and Parmley[5] and Wells.[14] Because the risk of coronary artery disease increases markedly with the number of risk factors,[1,3,15] nonsmokers with hypertension or hypercholesterolemia and exposed to environmental tobacco smoke are likely to be at even greater risk of developing cardiovascular disease. It is well known that the risk of coronary heart disease caused by voluntary smoking decreases by about half after 1 year of smoking cessation and after several years approaches that of people who have never smoked.[16] Similar health benefits should occur in previously environmental tobacco smoke–exposed nonsmoking individuals when environmental tobacco smoke is removed from the environment in which they work and live.[8]

Exposure to Environmental Tobacco Smoke

Although the proportion of smokers in the United States is decreasing, 32% of men and 27% of women aged 20 and older smoke cigarettes.[17] These smokers will expose a vast number of nonsmokers to environmental tobacco smoke, and it has been estimated that approximately 50 million nonsmoking adults over age 35 are regularly exposed to environmental tobacco smoke.[17] Additionally, we estimate that 50% of all children live in families with one or more smokers. In a survey conducted in 1979–1980, 63% of nonsmokers reported being exposed to environmental tobacco smoke for more than 1 hour per week, 35% were exposed to environmental tobacco smoke for more than 10 hours per week, and 16% were exposed to environmental tobacco smoke for at least 40 hours per week.[18] It is likely that exposure of nonsmokers to environmental tobacco smoke has decreased in recent years because of the increased public awareness of the hazards of environmental tobacco smoke, increased restrictions on smoking areas, and better ventilation of the workplace. The public has now begun to understand the detrimental health effects of environmental tobacco smoke exposure, but this increased awareness has not eliminated exposure to environmental tobacco smoke of spouses and children living in a smoker's home or that occurring in some workplaces and public buildings.

Cardiovascular Effects of Environmental Tobacco Smoke

Environmental tobacco smoke produces acute effects on cardiovascular function in human studies. In subjects with stable angina, environmental tobacco smoke increases resting heart rate, blood pressure, and blood carboxyhemoglobin, and reduces the duration of exercise that induces angina.[19,20] Environmental tobacco smoke also produces adverse effects on the exercise performance of healthy people.[21] Several studies have found increases in the incidence of nonfatal heart disease, including angina and myocardial infarction, among nonsmokers exposed to environmental tobacco smoke.[22,23]

A few small sample cases show direct involvement between environmental tobacco smoke and peripheral vascular disease. For example, Bocanegra and Espinoza[24] reported Raynaud's phenomenon in two successive wives of a chain-smoker. The symptoms of both nonsmokers, as would be expected, subsided after they were no longer exposed to environmental tobacco smoke. Cigarette smoking is a major, preventable risk factor that promotes atherosclerotic peripheral vascular disease,[1,2] and it is likely that environmental tobacco smoke also increases the risk for peripheral vascular disease, although the latter hypothesis remains to be studied.

Mechanisms of Inducing Cardiovascular Disease

Nicotine, the drug in tobacco that causes addiction, produces acute increases in heart rate and blood pressure.[25] Cigarette smoking has been shown to increase platelet aggregation and cause endothelial cell damage.[26–28] Polycyclic aromatic hydrocarbons present in smoke (for example, benzo[a]pyrene) are capable of inducing and accelerating the development of atherosclerosis.[29,30] Exposure to environmental tobacco smoke will also increase carbon monoxide levels in red blood cells. Studies indicate that increased carbon monoxide levels in humans result in a more rapid onset of angina[31] and increased arrhythmias[32] in exercising nonsmokers. A recent study indicates that environmental tobacco smoke sensitizes circulating neutrophils in humans and may cause their subsequent activation and oxidant-mediated tissue damage, leading to carcinogenesis and atherosclerosis.[33] It is likely that these and more yet-to-be-identified mechanisms are involved in increasing the risk of heart disease in persons exposed to environmental tobacco smoke.

Potential for Prevention

Although regulation of tobacco products is specifically prohibited under the Federal Hazardous Substances Act, many actions have been taken to protect the health of nonsmokers. For example, cigarette smoking has been banned from air flights in the 48 contiguous states; and as of March 1991, laws restrict smoking in public places in 46 states, in public-sector workplaces in 38 states, and in private-sector workplaces in 17 states.[34] Many hospitals, health care facilities, and private and public workplaces are smoke-free. The benefit of restricting smoking in buildings and workplaces is obvious, but the effect of a greater awareness of the importance of reducing environmental tobacco smoke in the home has not been evaluated.

The final conclusion of the 1986 Surgeon General's Report was that separating the smokers and nonsmokers within the same air space may reduce but does not eliminate the exposure of nonsmokers to environmental tobacco smoke. Attempts to control tobacco smoke by increasing room ventilation can be futile, and the only sure way to protect nonsmokers from environmental tobacco smoke is to eliminate smoking from areas that they share with nonsmokers. Environmental tobacco smoke must now be considered an environmental toxin from which the public and workers should be protected. Thus, it is the responsibility of the employer to protect workers, and of public building managers, to protect the public from environmental tobacco smoke exposure. It is the responsibility of parents to ensure that their children are not exposed to environmental tobacco smoke in the home, and the responsibility of everyone to eliminate this health hazard from the environment.[5]

Summary

Although the number of cardiovascular deaths associated with environmental tobacco smoke cannot be predicted with absolute certainty, the available evidence indicates that environmental tobacco smoke increases the risk of heart disease. The effects of environmental tobacco smoke on cardiovascular function, platelet function, neutrophil function, and plaque formation are the probable mechanisms leading to heart disease. The risk of death due to heart disease is increased by about 30% among those exposed to environmental tobacco smoke at home and could be much higher in those exposed at the workplace, where higher levels of environmental tobacco smoke may be present. Even though considerable uncertainty is a part of any analysis on the health affects of environmental tobacco smoke because of the difficulty of conducting long-term studies and selecting sample populations, an estimated 35,000–40,000 cardiovascular disease–related deaths and 3,000–5,000 lung cancer deaths due to environmental tobacco smoke exposure have been predicted to occur each year.

The AHA's Council on Cardiopulmonary and Critical Care has concluded that environmental tobacco smoke is a major preventable cause of cardiovascular disease and death. The council strongly supports efforts to eliminate all exposure of nonsmokers to environmental tobacco smoke. This requires that environmental tobacco smoke be treated as an environmental toxin, and ways to protect workers and the public from this health hazard should be developed. According to a 1989 Gallup survey commissioned by the American Lung Association, 86% of nonsmokers think that environmental tobacco smoke is harmful and 77% believe that smokers should abstain in the presence of nonsmokers. However, programs aimed at further educating the public about the cardiovascular effects on nonsmokers of exposure to environmental tobacco smoke must be strengthened and remain a major component of the AHA mission. A smoke-free environment in the home, public buildings, and workplace should be the goal of society.

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Even a Little Secondhand Smoke Is Dangerous

Stanton A. Glantz, PhD
William W. Parmley, MD

As more and more nonsmokers have come to understand the dangers associated with breathing secondhand smoke,[1,2] the number of communities enacting ordinances requiring smoke-free workplaces and public places has increased rapidly. As of May 2001, hundreds of communities had enacted laws requiring smoke-free workplaces, smoke-free restaurants, and smoke-free bars. California requires all workplaces, including restaurants and bars, to be smoke-free.[3,4] The theme for the World Health Organization's World No Tobacco Day in 2001 was "clean indoor air" and communities throughout the world are beginning to clear the air of secondhand smoke. Not only do the laws protect nonsmokers from the toxins in secondhand smoke, but they also create an environment that helps smokers cut down or stop smoking.[5]

The tobacco industry's efforts to slow the spread of smoke-free environments has included a systematic effort to attempt to undermine the scientific evidence that passive smoking causes disease.[6-8] One common theme is that the dose of toxins a nonsmoker inhales is tiny compared with the dose the smoker receives, implying that the risks are trivial or nonexistent. Such statements are based on measuring the delivered dose of 1 or more of the 4000 chemicals in secondhand smoke. The problem with such calculations is they can be manipulated by selecting the particular constituent of smoke to be the one that has low absorption or rapid clearance.[1] The real measure of effect should not be the dose of one chemical or another, but rather the biological effect of breathing the secondhand smoke.

The article by Otsuka and colleagues[9] in this issue of THE JOURNAL adds substantially to the case that short-term passive smoking adversely affects endothelial function in ways that immediately compromise the cardiovascular system.[10] The investigators demonstrated that, in healthy young volunteers, just 30 minutes of exposure to secondhand smoke compromised the endothelial function in coronary arteries of nonsmokers in a way that made the endothelial response of nonsmokers indistinguishable from that of habitual smokers.

See also p 436.

The investigators measured blood pressure, heart rate, and coronary flow velocity reserve before and after administering adenosine triphosphate using transthoracic Doppler echocardiography of the left anterior descending coronary artery. This innovative noninvasive approach to measuring coronary endothelial function appears to be ideal in these individuals, who have no evidence of coronary disease. Significantly, these substantial changes in endothelial function were not associated with changes in heart rate or blood pressure.

Endothelial dysfunction may be at the heart of the development of atherosclerosis. Normal endothelial cells promote vasodilation and inhibit atherosclerosis and thrombosis, in part because of the release of nitric oxide.[11] Dysfunctional cells, on the other hand, contribute to vasoconstriction, atherogenesis, and thrombosis. Risk factors contribute individually to endothelial dysfunction and appear to be additive. One possible unifying hypothesis for the effects of risk factors is that they increase oxidative stress that mediates these effects.[12] Thus, reduction of risk factors improves endothelial function and reduces clinical coronary events. For example, in patients with hyperlipidemia, lipid lowering improves endothelial function both acutely[13] and chronically.[14]

The findings of Otsuka et al[9] are important not only because they illustrate the importance of preventing nonsmokers from any exposure to secondhand smoke, but also because they help to explain the relatively large risk of death and other cardiac events associated with passive smoking compared with active smoking. Passive smoking increases the risk of cardiac death or morbidity about 30%[15-21] compared with a doubling to quadrupling of risk associated with active smoking. Thus, the effect of passive smoking is as high as one third the effect of active smoking even though the dose of at least some of the constituents is much less than what the smoker inhales.[1]

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The first evidence that nonsmokers were sensitive to a mp[illegible]nt of tobacco smoke came from studies showing at sh[illegible]-term (30-minute) exposure to secondhand smoke tivated nonsmokers' platelets to nearly the extent that they re activated in smokers[22,23] and that passive smoking ineased the presence of endothelial cell morbidity in the ood.[23] These immediate effects on platelets probably act nergistically with the effects on endothelial function. The atelet effects convinced epidemiologists that the dosesponse curve for cardiovascular effects associated with tocco smoke exposure was not linear, but exhibited subantial effects at relatively low doses (at least compared with active smoker; the doses are high when measured against ther environmental toxins) that a passive smoker reeives.[18,20] In addition, animal studies demonstrated that exosure to the secondhand smoke from a single cigarette daily nduced atherosclerotic changes.[24] The fact that passive smokng does not induce additional effects in smokers[9,22] sugests that the underlying biochemical and cellular proesses saturate at the doses involuntary smokers experience.

While most people think of cancer when they think of active nd passive smoking, it is important to emphasize that heart lisease is also an important consequence of tobacco smoke xposure. This situation is particularly true for passive smokng; heart disease accounts for about 37 000 of the estimated 53 000 annual deaths attributed to involuntary smoking in he United States.[18] Another important difference between the effects of smoking on risk of cancer compared with risk of hea[illegible]sease is that the effects on cancer develop and resolve slowly (over a period of years) whereas the effects of smoking on the cardiovascular system occur rapidly.

The findings of the study by Otsuka et al[9] add to the evilence suggesting that everyone should be protected from even short-term exposure to the toxins in secondhand smoke. Communities should continue to require that workplaces, ncluding restaurants and bars, be smoke-free and mount public education campaigns to encourage smoke-free homes. Not only will everyone breathe better,[25] but they will also have healthier hearts.

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MEASURING SUSTAINABLE DEVELOPMENT

APPLICATION OF THE GENUINE PROGRESS INDEX
TO NEWFOUNDLAND & LABRADOR

THE ECONOMIC IMPACT OF SMOKE-FREE WORKPLACES:

AN ASSESSMENT FOR NEWFOUNDLAND & LABRADOR

Prepared by:
Ronald Colman, Ph.D, **GPI***Atlantic*
With research assistance from Jeffrey Wilson and Rob Rainer

March 2003

One analyst has further warned of the dangers of focussing exclusively on the economics of tobacco control, arguing that this allows the tobacco industry to deflect attention from the deadly health impacts of smoking and to shift the issue from the domain of public health to that of fiscal policy. Dr. Kenneth Warner of the University of Michigan maintains that any discussion of tobacco economics must always aim to *"force the issue of tobacco back where it properly belongs, in the domain of public health."*[7]

For these reasons, Part One of this study focuses on the broader health impacts of environmental tobacco smoke, which provide the basic rationale for the proposed legislation. Within that framework, Part Two then assesses the potential impact of the legislation on restaurant, bar and hotel sales in Newfoundland & Labrador.

1.2 The Precautionary Principle

It has now been explicitly recognized in international agreements, and in national and provincial legislation that lack of scientific certainty should never prevent decisive legislative action when there is strong evidence of potential severe or irreversible damage to human health and the environment. Indeed it is a flagrant and unethical misuse of science to invoke the need for incontrovertible evidence in order to delay action that could save lives.

Instead, the internationally accepted "precautionary principle" puts the burden of proof on those who argue, in the light of strong evidence to the contrary, that serious damage may *not* occur. For example, the Kyoto climate change accord is based on a consensus among the world's leading meteorologists on the International Panel on Climate Change that the weight of scientific evidence points to human-induced greenhouse gas emissions as a primary cause of potentially catastrophic global warming.

This does not mean that such evidence constitutes incontrovertible proof. Indeed, the climate change models on which these predictions are based are acknowledged to be imperfect. But the evidence is sufficiently strong and the danger of sufficient magnitude that a "precautionary" approach necessitates current cuts in greenhouse gas emissions. If the climate change models are proved wrong, then fossil fuels remain available to be burned with abandon.

The same principle applies to the scientific evidence on the health impacts of second-hand smoke and to legislation that can potentially protect citizens from serious and life-threatening illnesses.

Nearly 30 years ago, the U.S. Surgeon-General, Jesse L. Steinfeld, concluded that the very high carcinogenicity of cigarette smoke created a probable risk of lung cancer for nonsmokers.[8] It took 15-20 more years for that evidence to be scientifically validated beyond any reasonable doubt, and for leading scientific and health agencies throughout the world to confirm the causal link between environmental tobacco smoke and lung cancer.

These agencies include:

- The World Health Organization (1986 and 1999),
- The U.S. National Academy of Sciences of the National Research Council (1986),
- The Australian National Health and Medical Research Council (1987),
- The U.K. Department of Health and Social Security (1988),
- The U.S. Environmental Protection Agency (EPA) (1992),
- The U.S. Public Health Service (1986),
- The U.S. National Institute for Occupational Safety and Health (1991),
- The American College of Occupational and Environmental Medicine (1993 and 2000),
- The California Environmental Protection Agency (1997),
- The Australian National Health and Medical Research Council (1997),
- The United Kingdom Scientific Committee on Tobacco and Health (1998)
- The U.S. National Toxicology Program *(Ninth Annual Report on Carcinogens, 2000)*.[9]

These reviews, carried out by panels of respected, independent scientists or by government agencies with review by scientific expert panels, have all been scientifically rigorous and scrupulous in their methodologies and procedures.

For example, the California Environmental Protection Agency's comprehensive five-year study on the health effects of exposure to ETS was peer reviewed by California's Scientific Review Panel, a body created under California law to provide independent peer review of many scientific aspects of the state's toxic air contaminants and air pollution programs. The California EPA also held public workshops, solicited input from all interested parties including the tobacco industry, and made drafts of the report available for public comment and criticisms.[10]

In addition to the 12 official reports listed above, more than 40 scientific studies have now established the causal role of ETS in the induction of lung cancer. What is remarkable is the high degree of consensus that has emerged from all these published studies on the health hazards of second-hand smoke.[11] It is necessary to emphasize here both the scientific rigour of those studies and their broad agreement on the health effects of ETS because of the tobacco industry's consistent denials and because its strategy of choice has been to find fault with some aspect of each study's methodology.

1.3 The Tobacco Industry Case on "Scientific Certainty"

It has taken even longer for the evidence linking second-hand smoke and lung cancer to be translated into action designed to protect employees in particular and citizens in general from a known carcinogen. During this entire 30-year period, the tobacco industry has argued that the evidence was not conclusive and that workplace smoking bans were not justified in the absence of scientific certainty on the health impacts of second-hand smoke.

Assessing the consequences of such delay, Dr. David Burns, Division of Pulmonary Care and Critical Care Medicine at the University of California at San Diego Medical Center, notes:

> *"The scientific case against environmental tobacco smoke is now overwhelming. It is sobering to count the number of lung cancer deaths that might have been*

1: JAMA. 1993 Jul 28;270(4):490-3.

Comment in:
- JAMA. 1994 Feb 23;271(8):584-5.

Involuntary smoking in the restaurant workplace. A review of employee exposure and health effects.

Siegel M.

University of California, Berkeley/University of California.

OBJECTIVE--To determine the relative exposure to environmental tobacco smoke for bar and restaurant employees compared with office employees and with nonsmokers exposed in the home (part 1) and to determine whether this exposure is contributing to an elevated lung cancer risk in these employees (part 2). DATA SOURCES--MEDLINE and bibliographies from identified publications. STUDY SELECTION--In part 1, published studies of indoor air quality were included if they reported a mean concentration of carbon monoxide, nicotine, or particulate matter from measurements taken in one or more bars, restaurants, offices, or residences with at least one smoker. In part 2, published epidemiologic studies that reported a risk estimate for lung cancer incidence or mortality in food-service workers were included if they controlled, directly or indirectly, for active smoking. DATA EXTRACTION--In part 1, a weighted average of the mean concentration of carbon monoxide, nicotine, and respirable suspended particulates reported in studies was calculated for bars, restaurants, offices, and residences. In part 2, the relative lung cancer risk for food-service workers compared with that for the general population was examined in the six identified studies. DATA SYNTHESIS--Levels of environmental tobacco smoke in restaurants were approximately 1.6 to 2.0 times higher than in office workplaces of other businesses and 1.5 times higher than in residences with at least one smoker. Levels in bars were 3.9 to 6.1 times higher than in offices and 4.4 to 4.5 times higher than in residences. The epidemiologic evidence suggested that there may be a 50% increase in lung cancer risk among food-service workers that is in part attributable to tobacco smoke exposure in the workplace. CONCLUSIONS--Environmental tobacco smoke is a significant occupational health hazard for food-service workers. To protect these

workers, smoking in bars and restaurants should be prohibited.

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INSIGHTS: SMOKING IN WISCONSIN

A series of papers on Wisconsin tobacco use with recommendations for action, based on the 2003 Wisconsin Tobacco Survey of 8,000 Wisconsin adults.

Secondhand Smoke: Awareness, Attitudes and Exposure Among Wisconsin Residents

Series 2, Paper Number 4

UW-CTRI
Center for Tobacco Research and Intervention
University of Wisconsin Medical School

University of Wisconsin
Comprehensive Cancer Center

DHFS
The Wisconsin Department of Health and Family Services

EXECUTIVE SUMMARY

Since the 1986 U.S. Surgeon General's Report, *The Health Consequences of Involuntary Smoking*, first made Americans aware of the dangers of secondhand smoke, our understanding of the health consequences of environmental tobacco smoke for both nonsmoking adults and children has expanded greatly. In Wisconsin, secondhand smoke is estimated to cause 700 lung cancer and heart disease deaths each year and thousands more are made seriously ill by asthma, allergic attacks and infectious disease.[1]

The 2003 Wisconsin Tobacco Survey (WTS) interviewed over 8,000 Wisconsin smokers, former smokers and never smokers regarding secondhand smoke. This report summarizes the WTS findings and offers recommendations based on those findings. According to the WTS, Wisconsin residents agree that secondhand smoke is harmful, prefer smokefree environments (especially their homes and workplaces) and support of smokefree policies. In fact, approximately 94% of Wisconsin residents overall agree that secondhand smoke is harmful, including 88% of smokers. Younger residents (18-24 year olds) are more likely to find secondhand smoke dangerous than older Wisconsinites. Exposure to secondhand smoke occurs more often, however, among workers with less education, primarily those working in the service/hospitality and manufacturing sectors. These workers are more likely to support changes in smoking policies than those working in sectors where environments are more likely to be smokefree.

Survey respondents generally support smokefree environments, especially in the home and workplace. Respondents were very likely to restrict smoking in their **homes**. Close to 70% of Wisconsin households reported that they completely prohibit smoking. An additional 10% allow smoking in some places or at some times. Only three percent of respondents allow smoking at all times in their home.

Regarding **workplaces**, close to 75% of respondents believe that smoking should not be allowed in indoor work areas (including 55% of smokers). Black respondents preferred stronger workplace policies than Whites. Workers in the entertainment, lodging and recreation industries in particular supported stronger workplace policies on smoking.

While the past ten years has seen a reduction in exposure to secondhand smoke in the workplace, this reduction has not been uniform. Employees of restaurants, taverns and manufacturing facilities, as well as racial and ethnic minorities are more often exposed to secondhand smoke than other groups. Approximately twice as many respondents with a high school education or less were exposed to secondhand smoke in their workplace compared to those with a college degree or more.

Moreover, there was strong support for smokefree **restaurants**. More than 70% of respondents reported that they would support local laws making restaurants smokefree including three-quarters of nonsmokers and half of smokers. More women support smokefree restaurants than men as do more Blacks than Whites. Smokefree restaurants were also more attractive to respondents as customers. Half said they would be more likely to dine in a smokefree restaurant while only six percent said they would be less likely.

Finally, close to two-thirds of respondents supported policies that require **bars and taverns** to be either smokefree or only allow smoking in specific areas. This contrasts with the current situation where few bars are smokefree or have smoking restrictions.

[vii] **Author:** Siegel, M., University of California, Berkley,
Title: "Involuntary Smoking in the Restaurant Workplace."
Publication: Journal of American Medical Association 270
Year: (1993)
Page: 490-493.
Weblink:
<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8320789&dopt=Abstract>

; and **Author:** Center for Tobacco Research and Intervention, University of Wisconsin Medical School.
Title: "Insights: Smoking in Wisconsin; A Series of Papers on Wisconsin Tobacco Use with Recommendations for Action," based on the 2003 Wisconsin Tobacco Survey of 8,000 Wisconsin adults: "Secondhand Smoke: Awareness, Attitudes and Exposure Among Wisconsin Residents," Series 2, Paper Number 4
Year: (2005)
Page: 4

viii **Author:** U.S. Census Bureau, Current Population Survey, March 2000. Table P50.
Year: (2000).

Census Tract	Total Employed 16 and Older	Food Preparation and Serving	
		Number	Percent
145	4	0	0.0%
131	32	0	0.0%
116	176	29	16.5%
146	2,067	281	13.6%
150	296	32	10.8%
147	1,123	126	11.2%
141	381	40	10.5%
102	222	21	9.5%
138	275	33	12.0%
137	583	65	11.1%
121	250	8	3.2%
84	353	36	10.2%
100	192	10	5.2%
67	378	28	7.4%
132	355	25	7.0%
117	95	0	0.0%
113	780	58	7.4%
178	67	5	7.5%
83	320	7	2.2%
99	357	19	5.3%
115	101	11	10.9%
136	742	39	5.3%
66	803	42	5.2%
97	383	53	13.8%
81	426	59	13.8%
123	417	6	1.4%
134	953	77	8.1%
98	370	39	10.5%
86	377	51	13.5%
82	293	22	7.5%
135	911	76	8.3%
140	202	19	9.4%
89	405	30	7.4%
85	394	22	5.6%
118	180	0	0.0%
90	721	56	7.8%
148	842	61	7.2%
40	779	56	7.2%
104	185	0	0.0%
70	897	64	7.1%
87	403	30	7.4%
105	338	17	5.0%
149	878	85	9.7%
122	789	37	4.7%
88	646	41	6.3%
151	733	89	12.1%
154	154	0	0.0%
101	256	14	5.5%
69	629	33	5.2%
103	205	33	16.1%
63	814	26	3.2%
64	647	72	11.1%
155	428	45	10.5%
120	184	29	15.8%
110	2,043	143	7.0%
96	667	31	4.6%
12	818	50	6.1%
119	214	6	2.8%
47	1,350	152	11.3%
169	1,147	121	10.5%
65	764	58	7.6%
177	486	67	13.8%
166	756	155	20.5%
158	1,276	136	10.7%
68	744	65	8.7%
168	1,100	121	11.0%
108	1,481	148	10.0%
44	1,293	18	1.4%

Total for all Tracts:

15,524

167	1,202		148	12.3%
165	894		69	7.7%
46	963		83	8.6%
159	1,251		167	13.3%
156	411		50	12.2%
62	1,053		87	8.3%
139	307		34	11.1%
112	1,240		169	13.6%
91	1,011		69	6.8%
175	1,049		51	4.9%
45	1,018		74	7.3%
164	1,878		174	9.3%
176	932		96	10.3%
157	1,421		106	7.5%
20	845		47	5.6%
80	1,039		138	13.3%
106	346		25	7.2%
174	868		65	7.5%
107	995		105	10.6%
41	1,103		89	8.1%
179	1,402		103	7.3%
163	1,687		176	10.4%
11	1,180		82	6.9%
24	962		54	5.6%
19	1,335		106	7.9%
2.01	1,737		128	7.4%
42	1,060		53	5.0%
25	861		15	1.7%
188	623		68	10.9%
48	1,433		74	5.2%
21	1,076		49	4.6%
26	1,108		35	3.2%
180.01	608		57	9.4%
28	754		73	9.7%
187	1,187		94	7.9%
23	1,657		109	6.6%
18	1,305		63	4.8%
214	1,282		70	5.5%
71	927		112	12.1%
170	2,023		175	8.7%
39	1,258		43	3.4%
200	1,485		96	6.5%
133	474		74	15.6%
79	1,002		126	12.6%
51	1,467		41	2.8%
160	1,095		141	12.9%
22	735		15	2.0%
9	1,576		65	4.1%
77	2,784		279	10.0%
60	1,085		83	7.6%
43	2,140		122	5.7%
143	1,644		81	4.9%
162	986		92	9.3%
196	1,612		38	2.4%
130	968		34	3.5%
180.02	888		27	3.0%
36	817		24	2.9%
172	980		63	6.4%
111	1,137		54	4.7%
4	1,189		69	5.8%
201	1,394		89	6.4%
10	1,607		86	5.4%
61	1,002		62	6.2%
16	1,173		34	2.9%
78	2,415		263	10.9%
161	1,173		109	9.3%
173	1,440		114	7.9%
3.02	920		49	5.3%
14	1,032		75	7.3%
13	1,540		92	6.0%
29	853		27	3.2%
124	1,476		30	2.0%
72	1,618		69	4.3%
27	843		35	4.2%

34	2,688		111	4.1%
171	1,093		76	7.0%
49	2,062		90	4.4%
212	1,034		63	6.1%
54	1,669		86	5.2%
1	3,485		142	4.1%
17	1,959		79	4.0%
186	1,140		74	6.5%
15	1,385		85	6.1%
202	1,244		64	5.1%
184	837		46	5.5%
50	2,360		66	2.8%
31	1,575		63	4.0%
204	1,241		99	8.0%
191	1,582		90	5.7%
5.02	1,942		77	4.0%
52	722		20	2.8%
189	676		20	3.0%
33	2,201		132	6.0%
30	1,768		111	6.3%
37	1,153		21	1.8%
205	1,344		90	6.7%
35	1,423		65	4.6%
218	1,034		14	1.4%
57	1,383		43	3.1%
92	752		52	6.9%
109	3,598		198	5.5%
126	1,416		47	3.3%
206	1,857		75	4.0%
93	1,346		46	3.4%
213	589		6	1.0%
76	2,252		152	6.7%
59	1,772		88	5.0%
73	1,780		118	6.6%
203	1,624		45	2.8%
183	1,382		110	8.0%
58	2,000		83	4.2%
7	1,589		37	2.3%
194	1,921		98	5.1%
198	2,441		134	5.5%
5.01	1,663		48	2.9%
129	1,614		96	5.9%
128	1,464		64	4.4%
190	2,376		70	2.9%
185	873		32	3.7%
38	1,156		56	4.8%
6	2,767		123	4.4%
207	2,584		129	5.0%
209	1,461		64	4.4%
94	1,569		89	5.7%
192	1,586		65	4.1%
127	748		13	1.7%
144	1,710		77	4.5%
8	2,482		91	3.7%
32	1,210		53	4.4%
181	1,102		62	5.6%
193	1,300		39	3.0%
197	2,702		111	4.1%
208	1,922		113	5.9%
114	366		45	12.3%
199	1,828		124	6.8%
210	1,032		22	2.1%
55	1,617		54	3.3%
152	349		11	3.2%
211	804		34	4.2%
216	2,222		86	3.9%
125	1,131		43	3.8%
95	1,184		54	4.6%
53	1,053		38	3.6%
217	3,296		108	3.3%
56	1,115		24	2.2%
215	1,360		63	4.6%
195	1,808		60	3.3%
153	363		7	1.9%

3.03	1,039		52	5.0%
2.02	3,248		57	1.8%
182	1,097		46	4.2%
3.04	1,655		76	4.6%
3.01	890		6	0.7%
75	1,695		102	6.0%
74	1,820		230	12.6%
			15524	